Published in final edited form as:
J Allergy Clin Immunol. 2012 September ; 130(3): 657–662.e2. doi:10.1016/j.jaci.2012.06.024.

Sensitization and Allergic Histories differ between Black and White Pregnant Women

Ganesa Wegienka, PhD[1], **Christine LM Joseph, PhD**[1], **Suzanne Havstad, MA**[1], **Edward Zoratti, MD**[2], **Dennis Ownby, MD**[3], and **Christine Cole Johnson, PhD.**[1]

[1]Department of Public Health Sciences, Henry Ford Hospital, Detroit, MI

[2]Division of Allergy and Clinical Immunology, Henry Ford Hospital, Detroit, MI

[3]Department of Pediatrics, Georgia Health Sciences University, Augusta, GA

Abstract

BACKGROUND—Racial differences in allergic diseases have been reported with Black individuals suffering disproportionately compared to White individuals, although such studies have been more commonly done in pediatric populations.

OBJECTIVE—Determine whether there are differences in rates of allergic sensitization or prior diagnoses of asthma, hay fever or eczema in Black and White pregnant women.

METHODS—Women were recruited during pregnancy (regardless of allergic history) as part of a birth cohort study in the Detroit metropolitan area and were interviewed about prior doctor diagnoses of asthma, hay fever/nasal allergies/allergic rhinitis and eczema. Blood samples were collected, total IgE was determined and specific IgE measured for *Alternaria alternata*, cat, cockroach, dog, *Dermatophagoides farinae*, short ragweed, Timothy grass, and egg.

RESULTS—Black women (n=563) were more likely than White women (n=219) to have had at least one specific IgE ≥0.35 IU/mL (62.5% versus 40.2%, p<0.001). Black women had higher total IgE (geometric means 47.8 IU/mL (95%CI 42.5, 53.8 IU/mL) versus 20.0 IU/mL (95%CI 16.2, 24.6 IU/mL), Wilcoxon Rank Sum p<0.001). Black women were more likely to have had a prior doctor diagnosis of asthma (22.7% versus 16.0%, p=0.04) and eczema (21.9% versus 14.8%) but not hay fever (White 17.5% versus Black 15.7%, p=0.55). Associations persisted for total IgE, having ≥1 positive allergen-specific IgE and eczema after adjusting for common socio-economic or environmental variables.

CONCLUSIONS—Racial differences in allergic sensitization and diagnoses were present even after controlling for various factors. Future research should focus on prevention in order to ameliorate these disparities.

Keywords

pregnancy; IgE; atopy; asthma; eczema; allergic rhinitis; race; ethnicity

Corresponding author: Ganesa Wegienka, 1 Ford Place, 3E, Detroit, MI, 48202, Phone: 313-874-3566, Fax: 313-874-6730, gwegien1@hfhs.org.

No author has any financial relationships with a biotechnology and/or pharmaceutical manufacturer that has an interest in the subject matter or materials discussed in the submitted manuscript.

INTRODUCTION

Racial differences in allergic diseases (prevalence and severity) have been reported with Black/African American individuals suffering disproportionately compared to White individuals.[1–8] Few data exist to detail any racial differences in allergic diseases in adult women. A previous report demonstrated that women from minority groups in the Boston area tended to have higher total IgE and were more likely to be sensitized to aeroallergens than White women in the area. [6] The women from minority groups were also more likely to have had a prior asthma diagnosis, but were less likely to have had a prior diagnosis of hay fever or eczema than White women. [6] The authors also reported that race was more strongly associated with the allergic diseases than were current social economic status (SES) indicators from census group blocks where the women resided including income and education. However, few individual level variables were considered in these analyses and by design, the study population was enriched for women with allergies and asthma.

Using our cohort of women recruited during pregnancy for longitudinal study of their children, our goal was to examine whether pregnant Black and White women living in the same region (urban and suburban Detroit, Michigan) were sensitized to the same allergens at similar rates and whether the groups had received prior doctor diagnoses of asthma, hay fever and eczema at the same rates. In these epidemiological analyses, we also compared levels of total IgE between these groups and within subgroups based on prior doctor diagnoses. We then explored whether any observed differences could be explained, either fully or in part, by various components of current SES or other environmental factors measured at the individual level.

METHODS

Study Population

These analyses utilized an NIH and institutionally funded cohort study, the Wayne County Health, Environment, Allergy and Asthma Longitudinal Study or “WHEALS”, that enrolled pregnant women receiving care at Henry Ford Health System (HFHS) obstetrics clinics in urban and suburban Detroit, Michigan. The primary objective for the original cohort study was to examine early life exposures related to childhood allergic diseases in the offspring of the enrolled pregnant women. These pregnant women serve as the source population for these secondary analyses. WHEALS includes interview data about the women during pregnancy, coincident with several postpartum home visits and during a clinic visit for her child at age 2 years. Most women provided a blood sample during pregnancy at the time of enrollment in the obstetrics clinic. Dust samples were collected at the home visits and analyses of these data are incorporated into the present analyses. We have previously published details of the cohort’s creation. [9,10]

The last WHEALS birth was in December 2007 after enrolling 1258 pregnant women. Of the 1258 women enrolled, 290 identified themselves as “Caucasian/White” and neither Hispanic nor Middle Eastern (hereafter referred to as White) and 777 self-identified as “African American/Black” (hereafter referred to as Black). We collected 219 (75.5%) and 563 (72.5%) blood samples from the White women and Black women, respectively. Blood sample collection during pregnancy, which was not part of the original hypotheses of WHEALS, was not attempted for all women enrolled as financial resources did not permit the collection and processing of blood samples for all women. Thus, women who were not asked to provide a blood sample during pregnancy were recruited during a specific time period and their lack of a sample was not tied to any maternal or clinical characteristics. The women included in the present analyses have results on total IgE and allergen specific IgE

(sIgE). Women enrolled in WHEALS who were included and not included (no blood sample) in the analyses are compared to assess differences.

Data Collection

The baseline interview at recruitment during pregnancy included questions about maternal marital status, educational status, household income, smoking status and whether or not she paid for housing (made monthly rental or mortgage payments). The interview also included the following questions that serve as the source of outcomes for the analyses: “Did a doctor ever diagnose you with hay fever, nasal allergies or allergic rhinitis?”; “Did a doctor ever diagnose you with asthma?”; and, “Did a doctor ever diagnose you with eczema or atopic dermatitis?”. “Current asthma” was defined as ever having a doctor’s diagnosis of asthma plus having an asthma attack and/or taken asthma medications in the last 12 months.

The woman’s address at recruitment was used to determine whether the residence was “urban” (Detroit, Hamtramck or Highland Park) or suburban (all other settings). As part of the WHEALS protocol, dust samples were collected from their children’s bedroom floor in the first year of life and the samples were analyzed for cat allergen (Fel d 1), dog allergen (Can f 1), cockroach allergen (Bla g 2) and endotoxin using methods we have previously published.[11] No dust samples were collected during pregnancy and the samples collected postpartum were used as an approximation of the prenatal dust.

Blood was collected to determine levels of total and allergen-specific IgE [sIgE: dust mite (*Der f*), dog, cat, Timothy grass, ragweed, *Alternaria alternata,* egg and German cockroach (*Blatella germanica*)]. Measurement of total IgE and sIgE was performed according to the manufacturer’s standard protocols using the Pharmacia UniCAP system (Pharmacia-Upjohn Diagnostic Division, Kalamazoo, Michigan, USA). A positive sIgE was defined as ≥0.35 kU/l.

Statistical Analyses

Analyses were performed with SAS (Cary, NC, v9.2) and $p<0.05$ was considered statistically significant. Likelihood ratio chi-square tests and t tests were used to compare characteristics of participants who were included and excluded from the analyses. Percentages (positive sIgE; doctor diagnosis of hay fever/nasal allergies/allergic rhinitis, asthma and eczema; current asthma) and geometric means (total IgE) were used to describe the frequency of diseases and distributions of total IgE. Chi-square and Wilcoxon Rank Sum tests were used to compare the frequency of diseases and distributions of total IgE between Black and White women. A Jonckheere-Terpstra test for trend was used to compare the distribution of the total number of allergens to which the women were sensitized. Logistic regression models were used to investigate binary outcomes (at least 1 positive sIgE, doctor diagnoses, current asthma) and linear regression was used to model continuous data (log transformed total IgE) to determine whether SES and other individual-level factors could explain any observed racial differences. Data were transformed as needed to meet model assumptions. Change in effects criteria of 20% were used to evaluate confounders.

No single definition of SES has proven to explain racial disparities in allergic diseases. We used numerous factors to represent SES in our analyses including: household income, education and whether the woman paid for housing. We also considered additional factors not necessarily considered SES markers in the analyses including whether the woman was exposed to indoor pets during pregnancy, maternal age and marital status (was she married/living as married or not). We included additional models restricted to women who had dust samples with individual adjustments for the samples (endotoxin, dog allergen, cat allergen,

cockroach allergen) and for simultaneous adjustment for all variables including results from the dust analyses.

Our analyses considered how the association between race and the diseases varied in models with the addition of indicators of SES and other factors. Since our focus was on the association between race and the outcomes, our modeling strategy was to assess the change in the association (coefficient) for race for each outcome (allergic disease or total IgE) from the unadjusted model to models that were first adjusted for each potential confounder individually and then all potential confounders simultaneously. We could not adjust for whether the woman lived in an urban or suburban location as 140 (24.9%) Black women lived in a suburban setting and 89% of White women lived in a suburban setting; thus race was highly correlated with residence setting in our data. This correlation reflects the metropolitan Detroit area's demographic patterns.

RESULTS

Among White women who enrolled, those who provided a blood sample were no different ($p>0.05$) than those who did not with respect to any of the study characteristics, including number of prior pregnancies, income, education, payment for housing, indoor pet keeping during pregnancy, marital status, smoking status, urban residence or age at enrollment (Online Table I). Black women included in the study had higher incomes than Black women not included (Chi square $p=0.009$).

Among women included in the analyses, White women had fewer prior pregnancies, greater income and education, and were more likely to make payments for housing than Black women. White women also were more likely to have indoor pets, have breastfed their child, be married or living as married, and live in the suburbs. The Black women tended to be younger (Online Table I).

sIgE

Black women were more likely to have had at least one positive specific IgE ≥0.35 IU/mL compared to White women (62.5% versus 40.2%, $p<0.001$) (Table I). Compared with White women, Black women in WHEALS were statistically significantly more likely to be sensitized to each of the specific allergens (dog, cat, *A. alternata*, cockroach, short ragweed, Timothy grass) with the exception of dust mite (Chi square test $p=0.86$) and egg (Chi square test $p=0.37$) (Table I).

Overall, Black women were sensitized to more allergens than White women (test for trend, $p<0.01$) (Online Table II). However, the number of total allergens to which the women had a positive sIgE were not different between Black and White women when the groups were limited to only those who had at least one positive sIgE (test for trend, $p=0.11$) (Online Table II). After adjusting for each SES and other factors individually and simultaneously, the difference in the odds of having at least one positive sIgE between Black/African American and White women persisted (Table II).

Doctor Diagnoses of Allergic Diseases

In our study population, the rates of doctor diagnoses of hay fever did not differ between White (17.5%) and Black (15.7%) women ($p=0.55$) (Table I). After adjusting for SES and other factors individually and simultaneously, there was no evidence of difference in the odds of having a doctor diagnosis of hay fever between Black and White women. (Table II)

Black women were more likely than White women to have ever had a doctor diagnosis of asthma (22.7% versus 16.0%, $p=0.04$); however, the rates of current asthma were similar

(Table I). The odds ratio (OR=1.55, 95% CI 1.03, 2.33) was reduced after adjusting for SES and other factors individually (Table II). The association no longer persisted after adjusting for each factor simultaneously (Table II).

Black women were also more likely to have ever had a doctor diagnosis of eczema (21.9% versus 14.8%; p=0.025) (Table I). Black women were still more likely to have had the diagnosis even after adjusting for each SES and other factor individually as well as simultaneously (Table II).

Total IgE

Compared with White women, Black women in WHEALS had higher total IgE at enrollment (geometric means 47.8 IU/mL (95%CI 42.5, 53.8) versus 20.0 IU/mL (95%CI 16.2, 24.6 IU/mL), Wilcoxon Rank Sum p<0.001) (Table I). Based on a linear regression model, the unadjusted regression coefficient (beta) for race in the model of log transformed total IgE was 0.87 (95% CI=0.64, 1.10). This suggests Black women have a total IgE level that is approximately 139% higher than their White counterparts.

Adjusting for any individual factor or all factors simultaneously did not appreciably alter this association (Table III). The regression coefficient (beta) should be interpreted as the ($e^{beta} - 1$) percent increase in the mean total IgE for Black women compared to White women. All regression coefficients for race were p>0.05. We also compared total IgE between Black women and White women within subgroups defined by whether they: were positive for at least one sIgE, had a doctor diagnosis of asthma, had current asthma, had a doctor diagnosis of hay fever, and had a doctor diagnosis of eczema (Table IV). Regardless of whether the women had at least one positive sIgE or a prior diagnosis, Black women still tended to have higher total IgE than White women (all p<0.05) except for women who had current asthma. While the total IgE geometric mean was higher for African American women compared with White women with current asthma, the difference was not statistically significantly different (p=0.83) (Table IV).

DISCUSSION

In our cohort of pregnant women in the urban and suburban areas of metropolitan Detroit, Black women had higher total IgE than White women, regardless of whether or not they were sensitized to allergens or had prior allergy-related diagnoses. In addition, Black women were more commonly sensitized to at least one allergen than White women and were more likely to have ever had a doctor diagnosis of eczema. In our population, there were no specific allergens to which White women were more commonly sensitized than Black women. None of the observed differences could be explained by current SES or other concurrent factors.

Our analyses, and preliminary data from some of these women previously published in an analysis of genetic ancestry,[12] demonstrate that racial differences in allergic sensitization exist in pregnant women in our region and Black women are disproportionately affected compared to White women. In their study of 882 pregnant women (169 were Black and 577 were White) in the Boston, MA area, Litonjua et al. reported higher total IgE for Black women compared with White women but White women were more likely to have reported a doctor told them they had hay fever (44.2% versus 27.9%) and eczema (22.8% versus 17.8%).[6] They also reported that Black women were more likely than White women to have been told by a doctor that they have asthma (50.9% versus 27.9%), as well as be sensitized to each of the allergens examined (dust mite, dog, cat, cockroach, *Aspergillus, Alternaria*, ragweed and ryegrass). Black women were approximately 2.5 times more likely than White women to be sensitized to ≥3 allergens. The racial differences were not explained by census

block group-level SES variables. The overall sensitization and diagnosis rates for women in their study were higher than the rates in our study for both Black and White women. In contrast, Black women rather than White women were more likely to have a prior eczema diagnosis in our study and differences in asthma rates between Black women and White women in our study did not remain statistically significant after adjustment. However, our study population was not enriched with women reporting an allergic history as the Boston-area study was. Also, census block group-level rather than individual level data (as in our analyses) were used in the Boston study to adjust for differences in the groups. In our analyses, the associations between race and the allergic diseases persisted even after adjusting for these common SES and other variables reported by the women.

Surprisingly, few other data have been published on racial differences in allergic diseases in adult women. While not limited to pregnant women, NHANES III data indicated that Black individuals ages 6–59 years were more likely than White individuals to have had a positive skin prick test to at least one of ten allergens (adjusted OR=1.6, 95% CI 1.4, 1.9). [1] Data from NHANES 2005–2006 also demonstrated that total IgE was higher in Black/African American individuals than White individuals. [13] Based on data from individuals of all ages in the National Health Interview Survey, females (9.3%) had higher current asthma prevalence than males (7.0%) and Black individuals (11.1%) had higher rates than White individuals (7.8%).[14] Another example is from NHANES data (2001–2002 and 2003–2004) for which McHugh et al. reported that 14.0% (95% CI 12.3. 15.9) of White women and 13.5% (95% CI 11.2, 16.2) of Black women ages 20–85 years in their study had a prior asthma diagnosis. [15] Because risks for allergic diseases, including sensitization, may not be uniform across groups within a larger population, further insight is provided when associations are examined in subgroups (defined by characteristics such as race, gender and age) as we have done in our population of Black and White women of reproductive age.

We understand that diseases such as eczema, asthma and allergic sensitization may more commonly be concordant in children than the results we see in our analyses. However, racial disparities in these outcomes have not been well studied in women of reproductive age. Our results could be due to a combination of factors. For example, it is often believed that the majority of childhood asthma is attributable to allergic sensitization. This may not be true of asthma in adult women. Also, there could be racial differences in medical care treatment seeking behaviors in our participants which could lead to differences in diagnoses. We did not capture information on this. These reasons provide further support for the need for a well-designed and executed study of racial disparities in adult women.

Our analyses focused on a large group of pregnant Black and White women from the same region. Although our data do not allow us to identify specific underlying causes of allergic disease, our goal was to examine whether previously reported patterns in group level variables also existed in our cohort that included individual level data. Ideally, we would have examined the associations between race and the allergic disorders separately for urban and suburban residents; however, this was not possible. This study is cross-sectional and uses data on current exposures and diseases; we could not establish that the SES or other variables preceded incidence or establish risk factors for sensitization in the women. Further, we did not have data on all environmental exposures such as airborne particulates and the dust samples were collected after the child's birth. Part of the analyses was based on sIgE levels and sensitization does not necessarily indicate that symptoms occur in the presence of the allergen. However, sensitization is important as it is upstream of clinically apparent allergy in the causal pathway. Despite these limitations, we think there are many strengths of our study such as inclusion of a large group of Black women that were not selected based on elevated risk for allergy. Our detailed data on individual variables and dust samples are additional assets to our analyses.

While research continues on racial disparities in treatment efficacy for allergic diseases such as asthma, investigation of sources of racial differences in prevalence and incidence of these allergic diseases has been minimal. What could explain these racial differences? As Lintonjua et al. pointed out, allergies are associated with higher socioeconomic status in European countries. [6] However, adjustment for SES and other factors in our study did not ameliorate all observed racial differences. While efforts to identify causation should be embraced, identification of modifiable factors is crucial. Further, total IgE is higher in Black women regardless of disease status. Thus, not only do we need to know what causes this racial difference in total IgE, but we also need to understand whether there are racial differences in the role total IgE may or may not play in the development of allergic diseases. We agree, as Litonjua et al. suggested, that gene-environment interactions, varying patterns of exposure to allergens and pollutants, and psychosocial stress may be sources of the racial differences. [6] Recently, vitamin D deficiency has also emerged as an important area of opportunity for research in allergic disease [16–25] and would be a logical pursuit given that Black women of reproductive age may experience vitamin D "hypovitiminosis" (25(OH)D ≤15 ng/mL) at 10 times the rate of their White counterparts (42.3 versus 4.2%)[26].

Racial differences in our large cohort-based cross-sectional study demonstrate that Black women were more likely than White women to have elevated allergen-specific IgE levels, higher total IgE and to have had a prior doctor diagnosis of eczema, but not hay fever or asthma (after adjustment of common variables). The associations between race and these diseases could not be explained by current family income, maternal education or marital status, family housing payment, prenatal exposure to indoor pets, being a smoker or a host of other factors. While further evidence demonstrating the degree of racial differences in allergic diseases emerges, development of novel intervention strategies is essential. Our own group has suggested that prevention may be as important as disease management if racial disparities in asthma are to be eliminated. [27] To truly eliminate racial disparities in allergic diseases, we need to seriously focus on designing studies to address causation and to identify modifiable factors. To better understand the sources of racial differences, longitudinal studies of racially, socially, economically and residentially diverse groups sufficient in size to allow subgroup analyses are needed (for example urban dwelling Black individuals versus suburban dwelling Black individuals versus suburban dwelling White individuals versus urban dwelling White individuals). With the limited progress that has been made in reducing racial disparities in allergic diseases, radical new hypotheses and approaches are needed to tackle this critical public health problem.

Acknowledgments

This work was funded by NIAID and the Fund for Henry Ford Hospital.

References

1. Arbes SJ, Gergen PJ, Elliott L, Zeldin DC. 2005. Prevalences of positive skin test responses to 10 common allergens in the US population: Results from the Third National Health and Nutrition Examination Survey. J Allergy Clin Immunol. 2005; 116:377–383. [PubMed: 16083793]
2. Akinbami LJ, Moorman JE, Garbe PL, Sondik EJ. Status of childhood asthma in the United States, 1980–2007. Pediatrics. 2009; 123:S131–S145. [PubMed: 19221156]
3. Center for Disease Control and Prevention. Morbidity and Mortality Weekly Report. Vital Signs: Asthma prevalence, disease characteristics, and self-management educations – United States 2001–2009. May 3.2011 60
4. McDaniel M, Paxson C, Waldfogel J. Racial disparities in childhood asthma in the United States: evidence from the National Health Interview Survey, 1997–2003. Pediatrics. 2006; 117:e868–e877. [PubMed: 16651291]

5. Stevenson LA, Gergen PJ, Hoover DR, Rosenstreich D, Mannino DM, Matte TD. Sociodemographic correlates of indoor allergen sensitivity among United States children. J Allergy Clin Immunol. 2001; 108:747–752. [PubMed: 11692099]
6. Litonjua AA, Celedon JC, Hausmann J, Nikolov M, Sredl D, Ryan L, et al. Variation in total and specific IgE: Effects of ethnicity and socioeconomic status. J Allergy Clin Immunol. 2005; 115:751–757. [PubMed: 15805994]
7. Shaw TE, Currie GP, Koudelka CW, Simpson EL. Eczema prevalence in the United States: data from the 2003 National Survey of Children's Health. J Invest Derm. 2011; 131:63–67.
8. Janumpally SR, Feldman SR, Gupta AK, Fleischer AB. In the United States, Blacks and Asian/Pacific Islanders are more likely than Whites to seek medical care for atopic dermatitis. Arch Dermatol. 2002; 138:634–637. [PubMed: 12020225]
9. Wegienka G, Havstad S, Zoratti E, Woodcroft K, Bobbitt K, Ownby DR, Johnson CC. Regulatory T Cells in Prenatal Blood Samples: Variability with Pet Exposure and Sensitization. J Repro Immunol. 2009; 81:74–81.
10. Wegienka G, Havstad S, Bobbitt K, Woodcroft KJ, Zoratti EM, Ownby DR, Johnson CC. Within-woman change in regulatory T cells from pregnancy to the postpartum period. J Repro Immunol. 2011; 88:58–65.
11. Ownby DR, Peterson EL, Williams LK, Zoratti EM, Wegienka GR, Woodcroft KJ, et al. Variation of dust endotoxin concentrations by location and time within homes of young children. Pediatr Allergy Immunol. 2010; 21:533–540. [PubMed: 20088861]
12. Yang JJ, Burchard EG, Choudhry S, Johnson CC, Ownby DR, Favro D, et al. Differences in allergic sensitization by self-reported race and genetic ancestry. J Allergy Clin Immunol. 2008; 122:820–827. [PubMed: 19014772]
13. Salo P, Calatroni A, Gergen PJ, Hoppin JA, Sever ML, Jaramillo R, et al. Allergy-related outcomes in relation to serum IgE: Results from the National Health and Examination Survey 2005–2006. J Allergy Clin Immunol. 2011; 127:1226–1235. [PubMed: 21320720]
14. Akinbami LJ, Moorman JE, Liu X. Asthma prevalence, health care use, and mortality: United States, 2005–2009. National Health Statistics Reports. Jan 12.2011 (32)
15. McHugh M, Symanski E, Pompeii LA, Delclos GL. Prevalence of asthma among adult females and males in the United States: results from the National Health and Nutrition Examination Survey (NHANES), 2001–2004. J Asthma. 2009; 46:759–766. [PubMed: 19863277]
16. Camargo CA, Rifas-Shiman SL, Litonjua AA, Rich-Edwards JW, Weiss ST, Gold DR, et al. Maternal intake of vitamin D during pregnancy and risk of recurrent wheeze in children at 3 years of age. Am J Clin Nutr. 2007; 85:788–795. [PubMed: 17344501]
17. Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers and cardiovascular disease. Am J Clin Nutr. 2004; 80:1678S–1688S. [PubMed: 15585788]
18. Lintonjua AA. Childhood asthma may be a consequence of vitamin D deficiency. Curr Opin Allergy Clin Immunol. 2009; 9:202–207. [PubMed: 19365260]
19. Lange NE, Litonjua AA, Hawrylowicz CM, Weiss S. Vitamin D, the immune system and asthma. 2009; 5:693–702.
20. Weiss ST. Bacterial components plus vitamin D: the ultimate solution to the asthma (autoimmune disease) epidemic? J Allergy Clin Immunol. 2011; 127:1128–1130. [PubMed: 21411129]
21. Ly NP, Litonjua AA, Gold D, Celedon JC. Gut microbiota, probiotics, vitamin D, asthma and obesity. J Allergy Clin Immunol. 2011; 127:1087–1094. [PubMed: 21419479]
22. Camargo CA, Clark S, Kaplan MA, Lieberman P, Wood RA. Regional differences in EpiPen prescriptions in the United States: the potential role of vitamin D. J Allergy Clin Immunol. 2007; 120:131–136. [PubMed: 17559916]
23. Staples JA, Ponsonby A, Lim L, McMichael AJ. Ecologic analysis of some immune-related disorders, including type 1 diabetes, in Australia: latitude, regional ultraviolet radiation, and disease prevalence. Environ Health Perpect. 2003; 111:518–523.
24. Mullins RJ, Clark S, Camargo CA. Regional variation in epinephrine autoinjector prescriptions in Australia: more evidence for the vitamin D-anaphylaxis hypothesis. Ann Allergy Asthma Immunol. 2009; 103:488–495. [PubMed: 20084842]

25. Rudders SA, Espinola JA, Camargo CA. North-south differences in US emergency department visits for acute allergic reactions. Ann Allergy Asthma Immunol. 2010; 104:413–416. [PubMed: 20486331]

26. Nesby-O'Dell S, Scanlon KS, Cogswell ME, Gillespie C, Hollis BW, Looker AC, et al. Hypovitiminosis D prevalence and determinants among African American and white women of reproductive age: third National Health and Nutrition Examination Survey, 1988–1994. Am J Clin Nutr. 2002; 76:187–192. [PubMed: 12081833]

27. Joseph CLM, Williams LK, Ownby DR, Saltzgaber J, Johnson CC. Applying epidemiologic concepts of primary, secondary and tertiary prevention to the elimination of racial disparities is asthma. J Allergy Clin Immunol. 2006; 117:233–240. [PubMed: 16461121]

Key Messages

- Racial differences in allergic sensitization and eczema in adult women require further research to identify causes and potential preventative measures.

Table I

Descriptive statistics and percents for baseline physician diagnoses, allergic sensitization and race.*

	White	Black	p-value†
N	219	563	
Total IgE Geometric Mean (95% CI)	20.0 IU/mL (95%CI 16.2, 24.6 IU/mL)	47.8 IU/mL (95%CI 42.5, 53.8)	<0.001
Had ≥ 1 allergen specific IgE ≥ 0.35 kU/l?	88 (40.2%) N (%)	352 (62.5%) N (%)	<0.001
Allergen specific IgE≥ 0.35 kU/l:			
Dog	32 (14.6%)	130 (23.2%)	0.008
Cat	38 (17.4%)	128 (22.9%)	0.097
Alternaria alternata	26 (11.9%)	150 (26.8%)	<0.001
Cockroach	12 (5.5%)	109 (19.5%)	<0.001
Dust mite (Der F)	50 (22.8%)	131 (23.4%)	0.86
Short Ragweed	45 (20.6%)	214 (38.3%)	<0.001
Timothy grass	38 (17.4%)	175 (31.4%)	<0.001
Egg	2 (0.9%)	12 (2.1%)	0.37
Doctor Diagnoses			
Ever had doctor diagnoses of hay fever/nasal allergies/allergic rhinitis	38 (17.5%)	87 (15.7%)	0.55
Ever had doctor diagnosis of asthma	35 (16.0%)	128 (22.7%)	0.037
Current asthma (ever doctor diagnosis plus either attack or asthma meds in last 12 months)	21 (9.6%)	48 (8.5%)	0.64
Ever had doctor diagnosis of eczema	32 (14.8%)	122 (21.9%)	0.025

*Totals vary due to some missing data: 16 missing doctor diagnosis to hay fever/nasal allergies/allergic rhinitis; 1 missing response to doctor diagnosis of asthma and currents asthma; and, 9 missing responses for doctor diagnosis of eczema.

†P-values test differences in percents of each outcome between Black women and White women

Table II

Logistic regression analyses (odds ratios with 95% confidence intervals) measuring associations between dependent variables and race (Black versus White) adjusted for covariates.

	≥1 positive allergen-specific IgE	Doctor diagnosis of hay fever/nasal allergies/allergic rhinitis	Doctor diagnosis of asthma	Doctor diagnosis of eczema
N				
Odds Ratio for Race (95% CI)	2.48 (1.80, 3.42) *	0.88 (0.58, 1.34)	1.55 (1.03, 2.33) *	1.62 (1.06, 2.48) *
Odds Ratio (95% CI) for race adjusting for each of the following individually:				
Number of prior pregnancies	2.64 (1.91, 3.66) *	0.87 (0.57, 1.33	1.56 (1.03, 2.36) *	1.53 (1.00, 2.36) *
Report of household income	2.69 (1.92, 3.77) *	0.94 (0.61, 1.46)	1.41 (0.92, 2.16)	1.79 (1.15, 2.78) *
Maternal education	2.59 (1.85, 3.62) *	1.04 (0.67, 1.62)	1.46 (0.95, 2.23)	1.71 (1.10, 2.66) *
Make monthly mortgage or rental payments	2.39 (1.74, 3.31) *	0.84 (0.54, 1.27)	1.52 (1.01, 2.31) *	1.70 (1.11, 2.61) *
Prenatal indoor pet(s)	2.61 (1.85, 3.69) *	0.88 (0.56, 1.38)	1.56 (1.01, 2.42) *	1.67 (1.06, 2.63) *
Mother was married or living as married during pregnancy	2.46 (1.75, 3.47) *	1.00 (0.64, 1.56)	1.24 (0.79, 1.92)	1.62 (1.03, 2.55) *
Women smoked	2.48 (1.80, 3.41) *	0.87 (0.57, 1.32)	1.56 (1.04, 2.36) *	1.62 (1.06, 2.47) *
Home is in urban setting	2.41 (1.63, 3.56) *	0.97 (0.58, 1.62)	1.38 (0.84, 2.26)	1.70 (1.02, 2.81) *
Maternal age at enrollment	2.45 (1.77, 3.39) *	0.94 (0.61, 1.43)	1.49 (0.98, 2.26)	1.66 (1.08, 2.56) *
Odds Ratio for Race (95% CI) adjusted for above variables †	2.59 (1.67, 4.03) *	1.02 (0.58, 1.82)	1.14 (0.66, 1.97)	1.73 (0.99, 3.04)
For women who had dust samples:				
Endotoxin N=583	2.61 (1.83, 3.74) *	0.81 (0.51, 1.28)	1.18 (0.76, 1.84)	1.79 (1.11, 2.89) *
Dog allergen N=611	2.78 (1.93, 4.02) *	0.80 (0.50, 1.28)	1.14 (0.73, 1.80)	1.74 (1.06, 2.84) *
Cat allergen N=610	3.07 (2.08, 4.53) *	0.90 (0.55, 1.47)	1.38 (0.86, 2.23)	1.93 (1.15, 3.24) *
Cockroach allergen N=604	2.83 (1.98, 4.05) *	0.80 (0.51, 1.26)	1.24 (0.80, 1.93)	1.77 (1.09, 2.86) *
Odds Ratio for Race (95% CI) adjusted for all variables including dust‡ N=564	3.35 (1.96, 5.73) *	1.20 (0.62, 2.34)	1.16 (0.61, 2.21)	2.31 (1.18, 4.55) *

* P<0.05

† Variables are: Number of prior pregnancies, income, education, rental/mortgage payments, pets in the home during pregnancy, whether the mother was married or living as married during pregnancy, woman was a smoker, urban residence and maternal age at enrollment.

‡ Variables are: Number of prior pregnancies, income, education, rental/mortgage payments, pets in the home during pregnancy, whether the mother was married or living as married during pregnancy, woman was a smoker, urban residence and maternal age at enrollment. Models also adjusted for levels of endotoxin and dog, cat and cockroach allergen from home samples.

Table III

The regression coefficient (beta) with 95% confidence intervals in the linear regression model for race (Black versus White) and log transformed total IgE. The crude estimate is included as are estimates adjusted for various SES variables and other selected factors.*

	Log transformed total IgE	Average % Increase in total IgE (for a Black woman compared to a White woman)
N	782	
Regression Coefficient (Beta) for Race (95% CI)	0.87 (0.64, 1.10)	139%
Regression Coefficient (Beta) (95% CI) for race adjusting for each of the following individually:		
Number of prior pregnancies	0.89 (0.66, 1.12)	144%
Report of household income	0.85 (0.61, 1.09)	134%
Maternal education	0.83 (0.59, 1.07)	129%
Make monthly mortgage or rental payments	0.87 (0.64, 1.10)	139%
Prenatal indoor pet(s)	0.86 (0.62, 1.11)	136%
Mother was married or living as married during pregnancy	0.84 (0.60, 1.09)	132%
Women smoked	0.88 (0.65, 1.11)	141%
Home is in urban setting	0.82 (0.53, 1.10)	127%
Maternal age at enrollment	0.85 (0.61, 1.08)	134%
Regression Coefficient (Beta) for Race (95% CI) adjusted for above variables †	0.78 (0.48, 1.09)	118%
For women who had dust samples:		
Endotoxin N=583	0.84 (0.59, 1.10)	132%
Dog allergen N=611	0.82 (0.56, 1.08)	127%
Cat allergen N=610	0.89 (0.62, 1.16)	144%
Cockroach allergen N=604	0.87 (0.62, 1.13)	139%
Regression Coefficient (Beta) for Race (95% CI) adjusted for all variables including dust‡ N=564	0.88 (0.52, 1.24)	141%

*Total IgE was log transformed to meet model assumptions. The regression coefficient (beta) should be interpreted as the ($e^{beta} - 1$) percent increase in the mean total IgE for Black women compared to White women.

†Variables are: Number of prior pregnancies, income, education, rental/mortgage payments, pets in the home during pregnancy, whether the mother was married or living as married during pregnancy, woman was a smoker, urban residence and maternal age at enrollment.

‡Variables are: Number of prior pregnancies, income, education, rental/mortgage payments, pets in the home during pregnancy, whether the mother was married or living as married during pregnancy, woman was a smoker, urban residence and maternal age at enrollment. Models also adjusted for levels of endotoxin and dog, cat and cockroach allergen from home samples.

Table IV

The geometric means (95% confidence intervals) of total IgE (IU/mL) within subgroups.

	White Women	Black Women	p-value*
At least 1 positive allergen- specific IgE	N=88 66.9 (52.0, 85.9)	N=352 93.8 (83.3, 105.5)	0.016
No positive allergen-specific IgE	N=131 8.9 (7.2, 11.0)	N=211 15.5 (13.3, 18.2)	<0.001
Doctor diagnosis of asthma	N=35 35.6 (18.1, 70.1)	N=128 78.5 (60.2, 102.5)	0.027
No doctor diagnosis of asthma	N=184 17.9 (14.5, 22.1)	N=435 41.3 (36.3, 47.0)	<0.001
Current asthma	N=21 47.7 (16.3, 139.7)	N=48 56.4 (36.8, 86.3)	0.83
No current asthma	N=198 18.2 (14.9, 22.2)	N=211 47.1 (41.6, 53.3)	<0.001
Doctor diagnosis of hay fever	N=38 33.6 (19.4, 58.1)	N=87 68.3 (51.5, 90.5)	0.033
No doctor diagnosis of hay fever	N=179 17.7 (14.2, 22.1)	N=466 43.8 (38.5, 49.9)	<0.001
Doctor diagnosis of eczema	N=32 29.2 (18.2, 46.9)	N=122 59.7 (45.4, 78.5)	0.013
No doctor diagnosis of eczema	N=185 18.8 (15.0, 23.8)	N=435 34.7 (30.8, 39.1)	<0.001

*Wilcoxon rank sum test.